blood-clot filling the subarachnoid space with no abnormality of the cord. One abnormal dilated vessel was found and tied off, and there was no further active haemorrhage.

Postoperatively the pain in the back disappeared, but she could move only her toes and regained no other active movement in the legs. When last seen in 1966 her blood pressure was over 300/120, and there were signs of inadequate blood supply to the legs. She was otherwise well, using a wheel-chair for ambulation.

COMMENT

The aneurysmal dilatations of collateral vessels in coarctation of the aorta are well documented in the literature (Boyd and Werblow, 1937 ; Cleland *et al.*, 1956). Histologically the walls of these dilated vessels are deficient in muscle and elastic tissue, and in some places they are composed solely of intima and adventitia (Cleland *et al.*, 1956). These changes may affect the spinal arteries as well as other collaterals. Wyburn-Mason (1943) recorded a total of seven cases in which enlarged tortuous spinal arteries with or without local aneurysm formation were found in cases of coarctation. Weenink and Smilde (1964) found a dilated anterior spinal artery at necropsy in a patient who presented with paraplegia of sudden onset.

The combination of spinal subarachnoid haemorrhage and coarctation of the aorta has, to my knowledge, been reported only once before (Wyburn-Mason, 1943). This patient had temporary weakness of one leg after the haemorrhage. Henson and Croft (1956), in their review of spinal subarachnoid haemorrhage, found permanent paraplegia directly due to the haemorrhage to be a rare occurrence.

Paraplegia with loss of pain and temperature and retention of touch and joint position sense is characteristic of the anterior spinal artery syndrome (Peterman *et al.*, 1958 ; Henneaux, 1960). The mechanism of its production in the above case must remain in doubt. The anterior spinal artery may have been compressed by clot. There may have been arterial spasm (Paulson, 1963), or, most likely, a portion of the cord previously supplied by the ruptured vessel became ischaemic.

My thanks are due to Associate Professor Solomon Posen and Mr. C. R. Letham for allowing me to see the patient and for their continuing interest.

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True Heartburn

Brit. med. J., 1967, **4**, 279–280

Heartburn, while sounding like a pathological description, is a descriptive term common in dyspeptic symptomatology—a symptom that most people experience and one that is not normally regarded with any great apprehension, nor does it carry a connotation of cardiac pathology. However, exceptions occur and the following case is one of them.

CASE REPORT

The patient, a bachelor aged 45 living with his mother, for many years had been a seaman, but during the previous two years had been working as a coffee roaster. On 7 January 1967 while sitting at home with his mother and feeling depressed as a result of delusions of persecution, he placed a poker in the fire and watched it heating until it was red. When he thought that the maximum heat had been generated on the tip of the poker he placed it over his heart and pushed hard. With little or no sensation of pain he observed that the poker passed quite easily into his chest ; he felt faint and fell on the floor. His mother summoned the neighbours for assistance, and, as it was seen that, though pale, he was still alive, the ambulance was summoned. He was admitted to the Western Infirmary in the early hours of 8 January.

Observation from a distance showed that the handle of the poker was projecting from the fourth left interspace, about 8 cm. from the midline (Fig. 1). Oscillation of the poker indicated that the heart was beating and the pulse rate was 120 per minute. His blood pressure was 80/50 mm. Hg and he was pale, perspiring, but not in any respiratory embarrassment. On closer observation it was seen that there was a ring of charring around the entrance wound, and that there was little or no haemorrhage. A lateral *x*-ray film taken with a portable machine confirmed what was already obvious, that the larger portion of the poker was in the left thoracic cavity.

Intravenous infusion was immediately begun and plasma administered, which produced a rise in systolic pressure to 100 mm. Hg. At this point it seemed prudent to explore the left thorax to determine the exact nature of the tissue damage. The chest was opened by an anterolateral incision through the fifth interspace. The lung was found to be collapsed and there was 200–300 ml. of blood in the thoracic cavity.

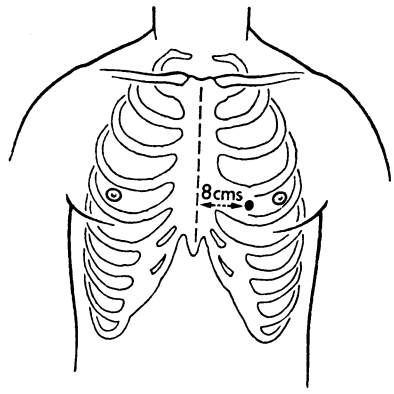

FIG. 1.—Entry wound in fourth left interspace.

Fig. 2 shows how the poker had passed through the pericardial cavity, the tip of the poker resting about 2 cm. from the posterior thoracic wall. There was no evidence of injury to the lung, but both entrance and exit wounds in the pericardium showed evidence of charring, and when the bridge of pericardium was divided it was seen that an area of the left ventricle, 2 by 4 cm., was whitened and clearly had been burnt as the hot poker passed over it. There was no active bleeding, and the poker was withdrawn without causing any further damage. Cardiac action seemed normal, and, as the patient's blood pressure had risen to normal levels after the transfusion of 2 pints (1,140 ml.) of blood, it was decided that no further action should be taken about the burning of the myocardium. As

a result of loss of tissue, the pericardium could not be sutured. A drain was inserted into the thoracic cavity and the thoracotomy wound was closed.

The first two days of his convalescence were marked by persistent hypotension, but tissue perfusion always seemed adequate and urinary secretion was established normally. There was some diminished air entry on the left side, and some evidence of collapse of the left lung. After these two days his convalescence progressed fairly satisfactorily. The main surgical complication of the convalescent period was a continued discharge from the front end of the thoracotomy wound, which was due to the establishment of an abscess cavity involving pericardial and thoracic cavities. With drainage this gradually closed, and, though one sinogram showed evidence of a bronchopleural fistula, the cavity closed without further complication.

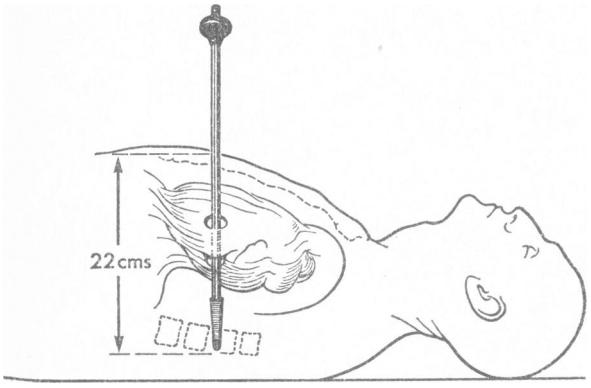

FIG. 2.—Position of poker in left hemithorax.

The electrocardiographic changes were particularly interesting. The first tracings after operation were consistent with a recent anterior myocardial infarction (Fig. 3). Widespread ST elevation was consistent with some degree of pericarditis. The most recent tracings (2 June) showed that ST elevation in anterolateral leads persisted. The absence of Q waves seemed to exclude the possibility of a ventricular aneurysm and screening of the chest also showed no evidence of an aneurysm. His clinical condition was excellent: he had no residual cardiac or respiratory symptoms; the anterior end of his wound had healed; and he had returned to his former occupation. His psychiatric state was investigated and the opinion was that he was suffering from a paranoid psychosis, though his suicidal act had apparently produced a remission in his mental symptoms. During his stay in the ward he was a model patient, and a casual observer would have found no abnormality in his mental attitude. He continued to attend as an outpatient for further psychiatric supervision. He had lost his delusions and acquired a myocardial scar.

COMMENT

There appear to be no references to similar cases in the literature. Rivkin (1963) and Tedeschi and White (1954) both reported on burning injuries to canine hearts in the course of studies designed to find out the damage caused to cardiac musculature by electric defibrillation. The lesions produced are similar to those that could be produced by myocardial infarction, but there seems to be no study determining the long-term behaviour of burnt areas of myocardium. In the present case the assumption is that the patient will behave as one who has had an anterior myocardial infarction. The prognosis should be good, as there is no occlusion of the main coronary vessels and it is likely that the anastomotic circulation is good. E.C.G. tracings do not suggest that the area of necrosis penetrated the full thickness of the myocardium, so it it not likely that he will develop an aneurysm. In a discussion with the patient it was not possible to find out why he chose this particular method of committing suicide except that the poker was handy; the actual idea of suicide did not occur until the poker was already heated and glowing red.

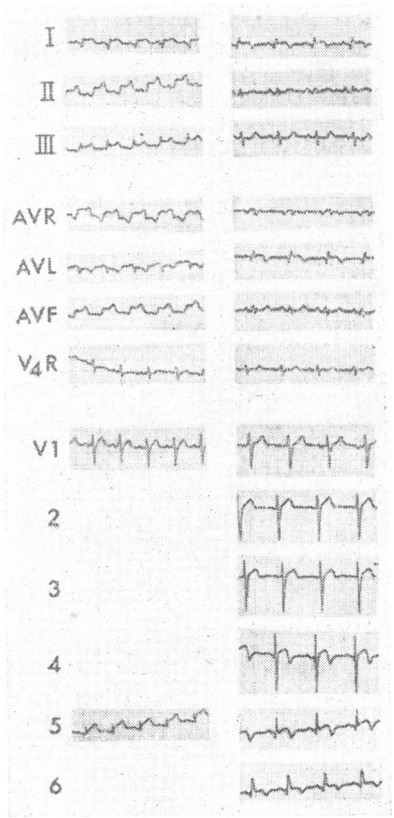

FIG. 3.—Electrocardiographic tracings; left, a day after injury (January 1967); right, six months later (June 1967).

This case is recorded as the first reported true case of acute heartburn, and also as an example of an avoidable cause of myocardial infarction.

My thanks are due to Dr. Ian Short and Dr. Gerald Timbury for their advice on the medical and psychiatric care of this case, and to the Department of Medical Illustration, Western Infirmary, for the illustrations.

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